limited to connective tissues and in which therefore the operative risk itself is slight.

1 "The Occurrence of a Positive Wassermann Reaction in Two Cases of Non-Specific Tumor of the Central Nervous System." Journal of the American Medical Association, January 6, 1912.

2 See a statement by Nonne in the Neurologisches Centralblatt, 1908, p. 751.

3 According to the diagram of Dejerine and Thomas, the second arch corresponds to the fourth dorsal segment; according to Gowers' diagram, it corresponds to the third.

4 Raven. Deutsche Zeitschrift für Nervenheilkunde, Vol. 44, p. 386.

5 Neurologisches Centralblatt, 1907, p. 520.

6 Bittorf and Schidorsky, Experimentelle Untersuchungen über das Wesen der Wassermann'schen Reaktion, Berliner Klinische Wochenschrift, 1912, No. 42.

THE VARIATIONS OF THE CLINICAL PICTURE OF MENINGEAL AFFECTION IN PULMONARY TUBERCULOSIS IN ADULTS. WITH CASE REPORTS.

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Whenever a case of pulmonary tuberculosis begins to show symptoms of meningeal irritation, the diagnosis of tubercular complication is generally predicted. No doubt this is as a rule correct; occasionally, however, one finds little at autopsy to verify such a diagnosis. The following case illustrates these statements and on that account should possess some interest.

The patient, G. V., male, age 30 years, had been suffering from pulmonary tuberculosis for three years, and at the time when first seen was in an "arrested" condition, doing a small amount of work, daily. One sister had died from tubercular meningitis, as a complication of pulmonary disease. No other facts of importance in the family history. The symptoms from which he demanded medical care came on suddenly. The patient was working in his garden when he began to suffer from intense frontal headache. This continued for several days when he began to vomit. At this time he presented the following: Patient was a large, well-formed, muscular man, slightly under weight, face flushed, pupils small, equal in size and sluggish in reaction to light and accommodation. There was no tenderness over the head, no rigidity of neck muscles, no history of syphilis, middle ear or sinus disease. Hearing and other senses normal. Mind perfectly clear. The knee jerks were absent and only a faint ankle jerk could be obtained. No changes in sensation. The pulse was slow (60 per minute), full and regular. Temperature 101° (10 a. m.). There was diffuse infiltration throughout entire right upper lobe with small cavity at apex. The lower lobe and middle lobe showed only slight thickening. There were few rales throughout these areas. The left upper showed signs of old fibroid condition with diminished resonance, and harsh breathing throughout with scattered fine rales. The right border of the heart was found about 1½ inches to the right of the sternum, apex normal, no murmurs. There was a slight amount of muco-purulent expectoration in which there were abundant tubercle bacilli. The abdomen was slightly retracted, no tenderness, stomach borders normal, no tumor or other pathological findings.

The headache was continuous, boring in character and located mostly in the frontal regions. The bowels were very costive. The patient retained but little food upon the stomach. The vomiting was almost "projectile" but not altogether so.

The patient was placed in a hospital under close observation, the 3rd day of his illness. The temperature varied from 97.2° f. in a. m. to about 101° f. p. m., pulse remained from 60 to 70 per minute. The bowels persistently refused to move even after large doses of salts, calomel, etc. Recourse was had to high colon flushings three times daily. Finally after three minims of croton oil in divided doses evacuation was secured. Morphine and chloral in large doses were the only drugs which seemed to relieve the headache.

The patient gradually became more and more restless and needed careful watching. Nevertheless his mind remained clear until shortly before death. Lumbar puncture was performed on the fourth dav of observation with the following results: The fluid flowed under considerable pressure, 15cc. was withdrawn; it was clear and limpid; albumen slightly increased, no polynuclear cells, but a slight increase in lymphocytes 16-20 per cu. mm., injection of fluid in peritoneal cavity of guinea pig gave positive results for tubercle bacilli. Ophthalmoscopic examination of eyes showed marked "choked disc" in both eyes.

After the lumbar puncture the patient's symptoms slightly improved; for two days he was able to take nourishment, and the headaches became less severe. Soon, however, they returned with greater intensity. Repetition of the puncture was refused. The neck muscles showed slight rigidity, the patient became unable to name objects presented to him, although he apparently understood what they were (paraphasia), was conscious of his surroundings. The knee jerks remained absent, the ankle jerk was slightly increased, and there was an uncertain Babinski. There was never any paralysis. Kernigs' sign became positive only a few days before death. His speech gradually became jumbled, although it was quite apparent that he knew what he wanted to say. The sphincter control was never lost or disturbed.

Urinalysis was entirely normal. The blood count showed only a moderate leukocytosis, the differential count was as follows: lymphocytes 25%, large mononuclears 5%, eosinophiles .5%, polynuclears 72.5. Total W. B. C. 10,000. No changes of note in the red cells. Hemoglobin Sahli 65%.

The patient gradually sank into coma, the lungs filled, became edematous, and death occurred nine davs after onset of the symptoms. A brief abstract of the post mortem findings follows.

On removing the skull cap the dura was densely adherent, the veins much dilated and markedly congested. The brain seemed to be markedly tense. There were slight fine adhesions over the entire cortex, and the pia showed marked edema and in places over the cerebrum fine thickenings and opacities. Nowhere were tubercles to be found. On sectioning of the brain the right ventricle was much dilated with fluid, but no other gross pathological changes were noted. The convolutions were everywhere well formed, and well developed. Microscopical examination of sections taken from the membranes and various regions of the brain, showed only slight pial infiltration and perivascular infiltration and thickening. There were present definite acute inflammatory changes but no areas of necrosis were to be demonstrated.

There were dense adhesive changes between the parietal and visceral pleura over both apices. The right lung showed multiple foci of tubercular infiltration throughout the entire upper lobe. The lesions on the whole were fairly well encapsulated. Scattered foci also appeared through the middle and lower lobes. The left upper lobe was also infiltrated throughout with scattered areas of fairly normal lung tissue. The lower left lobe was relatively clear. The bronchial glands were partly caseous and softened. Nothing of note was observed in the heart, liver and remaining organs.

Autopsy summary. Chronic pulmonary tuber-

culosis; edema of brain, with mild degree of acute inflammatory changes in the pia and cortical substance.

The interesting features from a clinical standpoint are: the sudden acute onset, the absence of very definite neurological signs of meningitis, knee jerks absent, late appearance of a Kernigs', prolonged clearness of the mind, late appearance of stiffness in the neck, as well as the occurrence of choked disc, slow pulse and complete absence of convulsions, paralysis, twitchings, photophobia and other usual signs of meningeal irritations. The pathological signs while slight in nature are enough to warrant the diagnosis of a mild meningo-encephalitis, due to the tubercular toxemia, but without actual tubercle formation.

That this mode of death in pulmonary tuberculosis in adults, certainly is not rare stands to reason in that I have observed three other cases develop suddenly typical symptoms of meningitis. The mode of onset, however, is particularly striking in these cases; it came on almost like an "eclampsia." The patients were all doing well, and supposedly in no danger. In one instance in a young girl of 18 years, the onset was with "hysterical" fits with marked emotional disturbance, there was a phantom tumor in the abdomen, the patient seemed dazed and unable to speak. Rapidly the picture cleared and the classical symptoms of meningitis asserted themselves. In another case, following a trivial quarrel with his sweetheart, a young man of 23 began to complain of severe headache, in 24 hours he was comatose, and in three days died with all the symptoms of meningitis. Many cases of tubercular meningitis can be traced to chronic middle ear disease but this factor did not exist in these cases. In another instance a young man of 21 years of age with a pneumonic type of phthisis began to show numbness of the finger ends and twitching of the index and middle fingers; in four days he was dead, having rapidly developed symptoms of meningitis.

In other cases there is a more chronic course with gradual progress, and in which the diagnosis from tubercular tumors is almost impossible. I observed one such case which at autopsy showed a solitary tubercle in the left optic thalamus.

It has long been known that one may get symptoms of meningitis in acute infectious fevers without very definite anatomical changes in the brain or membranes. Quincke is responsible for the statement that certain infectious diseases such as typhus, scarlatina, pneumonia and many febrile diseases cause a high pressure in the spinal fluid and sometimes increase the cellular content. In many cases the only theory available is the one of chemical toxin irritation. The French school have long held that the presence of lymphocytes in increased amounts in the spinal fluid indicates always meningeal irritation. Syphilis in any of its stages may cause spinal lymphocytosis and it is commonly found in paresis and locomotor ataxia. Noguchi's reaction should aid in differentiation between the syphilitic and non-syphilitic causes in explaining spinal lymphocytosis.

In a review of the literature up to 1904 Jessen quotes Schultze who made many observations showing that cases clinically showing symptoms of meningitis, anatomically gave no clue to their origin. Armand-Delille also claimed that the tubercular toxin could cause meningeal symptoms. Krannhols observed similar cases. In adults Kamboseff found tuberculous meningitis in only 2% of the cases. Kraemer in 477 tuberculosis cases found pial tuberculosis in 9.2%; in these, however, only one occurred in connection with phthisis. Therefore, Jessen states that tubercular meningitis develops very seldom in adults.

In a series of over 150 lumbar punctures done from a diagnostic standpoint, upon various nervous diseases reported in the *Journal of Nervous and Mental Diseases,* May, 1907, I observed several cases which showed an increase in lymphocytes in the spinal fluid, while at autopsy only very slight inflammatory changes were to be found microscopically. There is a point here to be remembered; if a patient who presents himself with symptoms of supposedly meningeal or cerebral origin has previously had syphilis, this alone will account for the lymphocytosis. Therefore from the presence of lymphocytosis alone we must not make a diagnosis of meningitis.

The exact source of infection of the meninges or brain in pulmonary tuberculosis seems to be uncertain. It may originate from the bronchial glands, but also direct from the lungs. Kraemer states that it may originate from the bronchial glands, joint tuberculosis, tubercular cervical glands, and also lung tuberculosis. Jessen states that tuberculous infection of the pia was first described by Von Dance in 1829. Buhl in 1859, before the discovery of the tubercle bacillus, thought that meningitis originated by the entrance into the blood of tubercular material. From the recent work we now know that tubercle bacilli frequently are to be found in the blood of pulmonary cases. The brain tissue in adults, however, seems to possess some inherent chemical resistance to invasion, as it seems to be but rarely attacked. Possibly the slow circulation, the presence of large amounts of blood, and the peculiar chemical consistency of brain tissue, containing as it does lecithin, etc., are directly destructive to tubercle bacilli. Experimental researches in part support this idea. But we can not explain the difference in the susceptibiity of the meninges in adults and children, except possibly on the grounds of developmental differences.

Kraemer in his material twice observed tuberculosis of the pulmonary veins. Oppenheim held that the infectious material gains entrance to the circulation through the pulmonary veins. Possibly the infection results from an infectious embolus. Strüpel states that the bacilli may travel through the lymph channels of the nerves to the arachnoidal sac of the cord and from there to the base of the brain. Leube felt satisfied that in one such case the mechanism of infection was upon

this basis. Peron concluded from his researches that the spread of the infection came through the cerebrospinal fluid. Possibly the bacilli passed from the lymph channels in the brain and tissues directly into the cerebrospinal fluid. The frequent presence of tubercle bacilli certainly show that they are present in such conditions but whether they pass into the fluid primarily to the formation of a localized lesion, or the lesion arises on the basis of their presence so far as one knows has not been solved.

Armand-Delille produced experimentally by ether and chloroform extracts of tuberculous material symptoms from the central nervous system, but only in a mechanical way hyperemia and leukocytic infiltration of the meninges. He never found a primary encephalitis, but only secondary symptoms from infiltration of the meninges.

O. Fischer analyzed 260 undoubted cases of tuberculous meningitis from the Leipsic Clinic. Fifty-seven cases occurred in patients over forty years of age; from forty to fifty years, eighteen cases. Most of the cases were of the basilar type, only in one-eighth of the cases was there inflammation of the convexity. Urine retention was a common symptom; rigidity of the neck muscles was common but not constant. The average duration was five to fourteen days, the longest seven months. None showed healing. Twenty-five lumbar punctures showed twenty-two times a plain lymphocytosis, only once were tubercle bacilli found. Two cases in the report are of unusual interest. A man in the last stages of heart disease was brought to the hospital "in extremis" and died of heart failure. There were no meningeal symptoms but necropsy showed a severe tuberculous meningitis. The other case was that of a man of forty-four years, with all the text-book symptoms of the disease including the "hydrocephalic cry"; at the necropsy there was advanced pulmonary and intestinal tuberculosis but no lesion whatever in the brain.

The disease seldom localizes itself upon the cerebral pia. Kraemer in forty-four cases found this condition only once. Seitz in fifty cases only once, Huebner in twenty-nine cases only once found meningitis of the cerebral area. One frequently finds a secondary infection of the brain substance and also of the pia spinalis. The inflammation of the pia spinalis is commonly found in connection with basilar meningitis.

Tubercle in the cerebral pia may follow direct trauma to the skull. Buol and Paulus describe such a case. A young man of twenty-eight who had recovered from an apical infiltration and had remained well for a year and a half had a fall in which he struck his head severely. In fourteen days he died of tubercular meningitis. At autopsy two small tuberculous masses were found in the central area of the convexity of the brain which were old and apparently healed, from these spread over the pia, generalized inflammation.

Vosnessensky reports a somewhat similar case in which operative treatment was attempted. A man of forty-five, some five years after a blow on the head, developed convulsions in the limbs on the left side, coming on at variable intervals. For four months previous, these crises occurred three times a day, and the muscles of the left wrist had been paralyzed for a month. Acute parietal headache had persisted for a month and difficulty in articulation for three days. Babinski was absent, there were no disorders in sensation, no disturbance of the pupils, though there was optic neuritis. After a few days the patient became comatose, with slow pulse, vomiting and dilatation of the pupils. Operation was performed over the right motor area, the dura was thickened and yellowish, adherent to the brain, and palpation disclosed a hard nodule. After incision of the cortex a firm yellowish mass two by one and three-fifths inches was removed. This was partly in the cortex and partly in the white matter. Death occurred the next day. Necropsy showed the growth to have been completely removed, there was no other in the body. Examination showed it to be a tuberculoma. Operations on such growths are still uncommon. Duret was able to collect but twenty-two on the cerebrum with nineteen cures and eleven on the cerebellum with nine deaths.

Psychic trauma or psychic excitement from varying causation seems also to be a predisposing factor in the production of tubercular meningitis. Continual worry, fatigue, the nervous constitution are mentioned by some authorities as predisposing causes. Zappert reports cases of hemiplegia, of tuberculous origin, due to a lesion in the internal capsule or over the convexity. Warfvinge reports cases of miliary tuberculosis with tubercles spread over the entire convexity, with marked exudate and pial thickening. The symptoms began with choreiform movements of right arm, leg and face, later the left side shows irritative symptoms.

Weintraud describes a case of meningitis of the convexity of the brain in a man of thirty-three with pulmonary tuberculosis. In this case he found also an endarteritis obliterans and thrombosis of the smaller vessels of the pia of tubercular origin.

Distinct motor symptoms may result from meningeal thickening over the motor centers. Matthes describes a case of meningitis in tuberculosis of the lungs, with paralysis of the hypoglossal nerve; at autopsy only meningeal thickening over the cortex, while the nucleus and nerve trunk were normal.

In general the prognosis of these cases is bad. Seldom does recovery ensue. Jirasek reports an instance of recovery, but such cases bring up the question that the disturbance may have been only toxic in origin. I have seen recovery ensue in the following case:

A young girl of 18 with severe destruction of the upper left lobe of the lung, developed rigidity of the neck muscles, weakness in the left arm and marked atrophy resulted in the muscles of the left forearm. The notes of the case are as follows:

Patient has been febrile (100°) daily and in bed some time. First began to suffer from rheumatic pains about the hips, both sciatic nerves tender to pressure, no loss of power in legs. Has

suffered from stiffness of the neck for several months; more on the left side. Would go away at times during the day. Some pain through back of head and neck at night. Would often be unable to turn head freely. Right wrist began to feel weak recently. Complains of soreness on left side. Felt a peculiar dullness or stiffness in the flexor group of muscles in the left forearm. Shoulder movements free and painless. Slight numbness in fingers and for days at a time lost almost completely muscular power in the left arm. At times she could not move the arm at all. The sensation to pain as tested by pin pricks was decreased on the ulnar side of the left arm up to the elbow but not on the radial side. Sensation was very dull on the little finger, and somewhat on the posterior surfaces of all the fingers, particularly the tips. Nodding of the head caused pain on the right side at the base of the skull. The muscles of the arm were slightly spastic particularly on forced movements. The reflexes were exaggerated. Neck muscles rigid on palpation particularly the left, muscles atrophic in left forearm, particularly the flexor group and also the ulnar, no joint disease anywhere. No tenderness over the vertebrae. Pupils unequal, left larger than right. Lung condition: marked destruction of left upper lobe; left border of heart beyond anterior auxiliary line, right lung fairly free.

It is interesting to note that shortly after these symptoms manifested themselves the patient developed tuberculosis of the left ankle joint, which was additional evidence of the wide dissemination of the disease. In spite of the severe lung condition the bone disease completely recovered and all the symptoms of the meningeal infection subsided. Tuberculin used throughout the course of the disease.

There is some question in this case whether the extreme destruction of the apex did not bring about a mechanical effect upon the left brachial plexus, but the slight symptoms on the right side are evidence against this theory.

This case illustrates that recovery may ensue in mild meningeal tuberculosis and unquestionably the use of tuberculin in this case was of great assistance. Dr. C. C. Browning has in an unpublished report described a marked case of basilar meningitis in a young girl treated with tuberculin with good recovery. This case was exhibited before the County Medical Society of Los Angeles.

P. Foa reports an instance of healing of a tubercle in the cerebellum. In a child which had died of parenchymatous nephritis, an old healed focus was found in the cerebellum. About a year previous symptoms of meningeal irritation had existed but passed away.

Janssen reports a case with meningeal symptoms which disappeared and the patient died of pulmonary tuberculosis. At autopsy several small tubercles were found in the meninges.

Freyhan reports a case of meningitis, which recovered in which tubercle bacilli were found. The acute type of basilar tuberculosis may terminate in a more chronic form. In fact it is a question in my mind whether the so-called cures are not merely the transformation into a latent condition. Clinically some signs will still persist. These cases may die at any time of an acute exacerbation of the disease. The tuberculous deposits become fibroid and encapsulated and give rise to Jacksonian epilepsy, contractures, asphasia, etc. Such a case is described by Anglade and Choevreaux. In a pulmonary case symptoms of epilepsy developed. After a number of years the patient died in status epilepticus. At autopsy there was besides a left sided lung tuberculosis, thickening of the pia, with normal cortex. In the area of the pial involvement, the vessels in the cortex were thickened, the cortical cells atrophic with necrotic patches and in places glial overgrowth. A solitary tubercle with bacilli was found over the right hemisphere in the meninges.

Von Dupre, Hauser and Sebilleau describe cases of chronic spinal meningitis, which showed at times acute exacerbations.

Spinal tuberculosis is even more rarely found than basilar. It is not unlikely that they are more often associated than we think only we do not make the necessary search. Isolated spinal meningitis of tubercular origin, however, has been described. Hoche reports a case of a twenty-six-year-old girl with both upper lobes infiltrated, the nervous system apparently normal, developed nightly delirium, opisthotonus, somnolence, weakness of the lower extremities and of the sphincters. Anatomically was found spinal tuberculosis of the meninges, and degeneration of the column of Goll and the pyramidal tract in the cord. Dinkler describes a case of motor paraplegia, disturbance of sensibility in the lower extremities and sphincter disturbance. Pressure symptoms also existed. The autopsy showed fungous outgrowth from the first and second dorsal vertebrae with compression of the cord and secondary myelitis and tuberculosis of the meninges.

Dupre, Hauser and Sebilleau describe a case with lung tuberculosis and pleurisy developed weakness of the legs with increased reflexes, positive Babinski reflex and stupor. Later delirium developed, disappearance of the reflexes, emaciation of the extremities, total anesthesia up to the navel; bed sores, and death in coma. The cord was affected from the sixth to tenth dorsal region, fibrous pachymeningitis (no tubercle bacilli), leptomeningitis thickening of pial vessels, and in the apendyma an old fibrous tubercle. In the gray substance, no changes. In the white substance, myelitis sixth to ninth dorsal segments. Meningeal thickening to sixth cervical segment. No caries of the vertebrae. In the Sylvian fissure of the brain on both sides, was found small old tubercle and meningo-encephalitis. The author held that the latter only caused psychic disturbance, and that the tuberculosis of the lungs and central nervous system existed for a long time and death came from the secondary infection in the spinal meningitis.

Other cases of mixed cerebral and spinal tuberculosis are reported by Von Londe and Brouardel.

Von Cerenville and Stilling report a case which showed marked degeneration of the column of Clark. In general one can say that tubercular meningitis spinalis may cause degenerative changes in the spinal cord also meningo-myelitis. Other etiological factors which seem to play a part are alcohol and syphilis.

It has long been known that nervous symptoms may arise in the course of familiar infectious diseases, such as typhoid fever without apparent anatomic changes in the meninges or in the brain. Definite meningitis and encephalitis also may be found after death without having given rise to any definite symptoms or no symptoms differing from the above group. The *Journal,* A. M. A., in an editorial recently calls attention to the work of Oseki in regard to these cases. Oseki found that in some instances, diagnosed as meningitis clinically, the post-mortem examination showed no gross changes except perhaps a moderate amount of edema and occasionally a very slight opacity of the membranes. On microscopic examination, however, there were found definite acute inflammatory changes with leukocytic and lymphocytic infiltration of the meninges and in the brain substance. The picture was that of an acute meningo-encephalitis, and as the process in the brain substance was most marked next to the meninges it seems reasonable to assume that the course of the process spread downward from the meninges. In another group of cases, however, the changes were confined entirely to the brain substance, showing that in encephalitis the meningeal symptoms may predominate as previously described by others. Oseki encountered cases of pneumonia in which meningitis and encephalitis were found to be present after death but of which there had been no symptoms whatever during life. In only one case could the changes be recognized by the naked eye.

Therefore it is very necessary to examine the cortex and meninges microscopically in order to detect the changes such as may exist. No doubt many of the cases of so-called "meningism" occurring in tuberculosis can be ascribed to the fact that in the absence of gross changes no microscopical study was made.

J. Finel and P. Gastinel in the April *Revue de Medicine,* review the autopsy findings in regard to a number of cases of meningeal involvement in the tuberculous. Extremely variable lesions from an acute inflammatory infiltration to diffuse sclerosis with fibrous nodules and cystic formations may be found. On the other hand the meninges may display a remarkable tolerance to the tubercle bacilli and the lesions run an entirely latent course. In other cases the meninges may be extremely intolerant and react with intense symptoms to minimal or transient injury. The phases of alternate latency and intolerance can only be explained on the basis of vaccination according to immunity theories.

Tuberculosis of the meninges in adults may, therefore, come on apparently with great rapidity and terminate within a few days. On the basis of these studies the suddenness of the onset in these cases may be explained in that there occurs a lighting up of an old focus, partially healed or latent. From a pathological standpoint this has been demonstrated in a few cases. The absence of definite anatomical findings in cases showing meningeal symptoms can be explained partly on the basis of insufficient microscopical examination of the brain and its coverings. In some well studied cases a mild meningo-encephalitis, without actual tubercle formation explains the symptoms. These changes, however, may occur without symptoms. Furthermore, actual tubercle formation in the meninges or brain substance may exist and give no hint of their presence. It is to be urged that tubercular patients presenting mental or nervous symptoms be carefully studied, and the pathologic-anatomic data secured.

Unquestionably, clinical healing of slight foci in the meninges or brain occasionally occurs. Tuberculin treatment should not be withheld in these cases, but the dosage should be much smaller than for the pulmonary disease. The anatomical evidence also demonstrated in the few cases accessible to study, that healing will take place. How long such lesions may remain "latent" and eventually cause death one can only judge in the light of similar conditions elsewhere.

From a predisposing standpoint, trauma to the skull seems important. Also psychic trauma certainly plays a factor.

The symptoms in these cases are often uncertain and present varying pictures. From the standpoint of the rather uncommon termination of pulmonary tuberculosis, the study of the varying causation, clinical picture and pathological anatomy of tubercular meningitis in adults holds forth considerable interest.

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TWO CASES OF APPENDICITIS WITH CERTAIN POST-OPERATIVE COMPLICATIONS.*

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The two cases that I have to report are both cases of acute appendicitis with rupture. Because of the not unusual complications that followed operation and the results obtained by measures instituted for their relief I consider them to be cases of special interest.

Case No. 1. The patient, a Japanese, 26 years of age, was first seen in consultation with Dr. von Adelung at time of entrance into hospital, August 1st, 1911. He complained of intense abdominal pain that had its onset three days prior. The pain was most severe in the lower right quadrant and was continuous. There had been no vomiting. There was marked tenderness on pressure over and about McBurney's point. Right rectus rigidity was very marked and on palpation a sense of tumefaction about the point of greatest tenderness could be elicited. Temperature was 100.6°; pulse 88; leukocytosis 24,200, 86% being polymorphonuclear.

The patient gave a history of obstinate constipation prior to his illness; appetite was always good and he said he never had suffered from any digestive disturbances. However, his general health had been poor for some time.

A diagnosis of acute appendicitis was made, and because of the rapidly increasing severity of the symptoms, the large area of marked tenderness on pressure, the tumefaction and high leukocyte count it was thought probable that rupture had taken place.

Immediate operation was advised and performed. Under ether anesthesia a Deaver incision was made and the peritoneal cavity opened. A moderate amount of sero-purulent fluid escaped and the presenting loops of intestine were red and presented areas covered with plastic exudate. No adhesions were encountered; nor was there any evidence of a protective wall around the appendix. The appendix was located with considerable difficulty. It protruded by about two-thirds of its length from behind the inner margin of cecum, high up, and was held firmly by its proximal extremity to the floor of the iliac fossa. It was dark red in color, greatly swollen, covered with particles of exudate and about its centre was a small gangrenous area where rupture had taken place.

Because of the indications of a well advanced peritonitis and the necessity for haste only that part of the appendix that protruded from behind the cecum was ligated with its mesentery and removed.

Multiple cigarette drains were used. The wound was left wide open for efficient drainage. Patient was returned to his bed with pulse of 86 and in fairly good condition. Fowler's position was resorted to and rectal infusion of normal saline 40 to 60 drops to the minute was commenced immediately. There was only slight nausea, no vomiting immediately after operation and six hours later he expelled considerable gas. For the next five days the indications were favorable for an uninterrupted recovery. The bowels were moved by enema with good result, the pulse ranged from 62 to 88 and the temperature 97 to 99; gas pains did not cause as much distress as usual.

On the fifth day the patient began to experience considerable abdominal distress in that there was pain, spasmodic in character, and increasing in intensity. He began to hiccough, felt nauseated and belched up gas and mucus occasionally. On the seventh day he was vomiting instead of belching, first stomach contents then a bile stained fluid. When the vomiting of the brownish fluid became quite frequent to the surprise of everybody three or four round intestinal worms were found in it. Up to the beginning of the ninth day the results of enemata became decreasingly small in quantity until finally they returned clear. Spasms of pain became intense, tympanitis very marked and peristalsis was visible during the paroxysms of pain. Vomiting continued, temperature in the afternoon was 97°, pulse 120; and the patient rapidly getting weaker. All symptoms pointed to obstruction of the bowels. Every effort had been made to overcome it but without avail, and a second operation was decided upon.

Ether anesthesia was employed, the original wound opened wide, and a rapid search was made for the obstruction. Loops of intestine were only slightly adherent, some greatly distended and others collapsed. One loop was found with a greatly distended proximal limb and a collapsed distal limb. It was quite adherent to the brim of the pelvis posteriorly. However, it was readily loosened with the finger and immediately the collapsed bowel began to fill with gas. This then was the cause of the obstruction. Drainage was again employed and the patient was again returned to his bed with pulse ranging from 130 to 150 and quite weak. Modified Fowler's position and the saline per rectum were used. Ten hours after operation the patient began passing large quantities of fecal matter involuntarily. For next thirty hours indications were very favorable, the pulse dropping down from 158 to 110 and the patient fairly comfortable Symptoms of intestinal obstruction then manifested themselves again. This time, however, they were much more acute and signs of prostration came on within a few hours. An emergency operation was then resorted to.

A distended loop of small intestine presented itself in the wound and in this was secured by means of purse-string sutures a quarter-inch rubber tube. Large quantities of gas escaped through it immediately, and with it came gushes of fecal matter. Within a few minutes the abdomen was much softer and the patient much relieved. Fifteen hours after the intestinal drainage was resorted to, pulse dropped from 140 to 110. Vomiting stopped at once and about twelve hours after the patient had a bowel movement of a large quantity of liquid fecal matter. Undoubtedly the obstruction was only partial but with the distension of the intestines from gas formation the kinking became more acute and the obstruction complete. However, with the release of pressure against the point of obstruction it became relieved and the intestinal contents began to go the normal way.

During the next sixty days patient improved rapidly. His pulse ranged about 80, was taking on weight, and suffered from no abdominal distress whatever. Bowels moved normally almost every day but occasionally enemas were given. The fistula required constant attention in that it discharged intestinal contents of great digestive power and as a consequence the skin around the wound would within a few hours, if neglected, show foci of ulceration. Granulations formed rapidly and it was hoped that the fistula would be closed by the natural contraction of the wound. An attempt was made to close it with a Lembert suture but without

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